CORRESPONDENCE

Two-year Clinical Outcomes of Stent Fractures Following Primary Femoropopliteal Stenting

In their article 'Incidence and the Clinical Impact of Stent Fractures after Primary Stenting for TASC C and D Femoropopliteal Lesions at 1 Year',[1] Davaine et al. explored the outcomes of patients following infra-inguinal stent fracture. They reported a 17.8% rate of stent fracture, but a remarkably low restenosis rate and negligible clinical impact at 1 year.[1]

These findings are at odds with our own experience. Our recently audited unpublished outcomes of primary femoropopliteal stenting over a 7-year period (441 stents in 263 limbs) demonstrate a stent fracture rate of 6.8% (30/441) with 66.7% and 88.9% restenosis at 1 and 2 years respectively. In all cases of restenosis, patients reported clinical symptoms of recurrent claudication or rest pain.

Femoropopliteal stent surveillance, as with infra-inguinal bypass graft surveillance, appears intuitive to maximise our patients' outcomes. However, both surveillance programmes are expensive, labour intensive, and are performed without any evidence base.[2] Daviane et al.[1] propose that although they feel these stent fractures are benign, a larger study should be performed to guide future therapy for these complications. Before we embark on such a study, perhaps it would be more valuable to look at the overall longer-term outcomes of femoropopliteal stenting beyond the traditional 1- and 2-year follow-up periods, and the surveillance regimens employed. We believe this would elucidate both the clinical value and economic benefit of surveying femoropopliteal stents. It would also support or refute this paper's conclusions that femoropopliteal stent fracture has low clinical impact.

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Stent Fracture: An Ongoing Story

Herein, Albayati et al. addressed several comments regarding our paper "Incidence and the clinical impact of stent fractures after primary stenting for TASC C and D femoropopliteal lesions at 1 year".[1] In their own experience they found a lower fracture rate (6.8% vs. 17.8%) and a higher in-stent restenosis (ISR) rate associated with stent fracture (66.7% vs. 40%) with consecutive symptoms in all cases.

Our analysis was on long lesions (220 ± 160 mm). Different studies have shown that the length of the stented segment and involvement of distal SFA and the popliteal artery were determinant factors governing higher stent fracture rates.[2] Moreover, long femoropopliteal occlusions were treated by the subintimal recanalization technique. The looped guidewire tip crosses the lesion in the subintimal space, hence splitting apart the media and significantly reducing the pool of smooth muscle cells available around the stent to induce intimal hyperplasia. In 2007, Ko et al.[3] reported better patency for revascularization of long femoropopliteal occlusions using the subintimal technique rather than the intraluminal technique. If Albayati et al. had narrowed their analysis to the most severe (TASC C and D) lesions with popliteal stenting, they would have probably observed results less discordant to ours. Furthermore, if not sought prospectively and systematically, fractures, and in particular minor fractures, are very often ignored and stent fracture rate underestimated.

Before correlating stent fracture to symptoms, we first should establish a link, which is not so obvious, between fractures and ISR or thrombosis. For instance, the correlation between a diffuse ISR and a focal fracture is still difficult to conceive. In contrast, does a focal ISR result from a stent fracture or from external and internal stresses (calcification, mechanical stresses, etc.)? Noteworthy, in human coronary arteries, only stent fractures with separation impact on the clinical outcome.[4] Hence, a larger study with higher statistical power is indeed required to determine more precisely where fractures occur and which type of fracture needs a reintervention.

We agree that long-term follow-up is mandatory. However, most ISR occur during the first 12 months;[5] thus, 2 years' follow-up seems sufficient. Furthermore, the stent is embedded into the arterial wall and the femoropopliteal disease is related to de novo lesions rather than restenosis. The 30 months results of our analysis, which should be shortly submitted, are in line with this notion.

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